



NOTES

ADRENAL HYPERFUNCTION

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Overproduction of $\geq$ one adrenal hormones
→ complex systemic disorders

CAUSES

- Pituitary/adrenal endocrine tumors
- Idiopathic, iatrogenic
- Increased aldosterone → hyperaldosteronism
- Increased cortisol → Cushing syndrome

SIGNS & SYMPTOMS

- Diffuse systemic symptoms due to systemic endocrine effects

DIAGNOSIS

DIAGNOSTIC IMAGING

CT scan/MRI

- Image tumors

LAB RESULTS

- Blood/urine tests; measure hormone levels

TREATMENT

- See individual disorders

CONN'S SYNDROME

osms.it/conns-syndrome

PATHOLOGY & CAUSES

- Type of primary hyperaldosteronism

CAUSES

- Adrenal glands produce too much aldosterone due to benign tumor (adrenal adenoma)
 - Forms in zona glomerulosa in adrenal gland

RISK FACTORS

- Individuals who are biologically female, 20–60 years old, with family history

SIGNS & SYMPTOMS

- Headache, facial flushing (due to hypertension)
- Constipation, muscle weakness, arrhythmias (if severe, due to hypokalemia)
 - Low potassium, high blood pressure (BP); unresponsive to treatment

VISIT...

LANZAROTE
Caliente.COM

DIAGNOSIS

DIAGNOSTIC IMAGING

CT scan

- Abdominal CT scan to differentiate tumor from idiopathic hyperaldosteronism

LAB RESULTS

- Adrenal vein sampling (CT scans do not detect lesions < 1cm/0.39in)

TREATMENT

SURGERY

- Unilateral adrenalectomy



Figure 11.1 The gross pathological appearance of an adrenal cortical adenoma in an individual with Conn's syndrome.

CUSHING'S SYNDROME

osms.it/cushings-syndrome

PATHOLOGY & CAUSES

- Endocrine disorder caused by **increased cortisol**
- Can be endogenous (caused by cortisol production inside body)/exogenous (iatrogenic)
- **Pseudo Cushing's syndrome**: estrogen-containing oral contraceptive pills → increased cortisol-binding globulin → increased total cortisol
 - Active hormone total free cortisol levels found in a 24-hour urine sample normal

CAUSES

Primary

- Tumor in zona fasciculata of adrenal gland secretes cortisol
- Adenoma (benign)/adenocarcinoma (malignant)

Secondary

- Iatrogenic

- Glucocorticoid steroid medications to treat inflammatory, autoimmune disorders (e.g. asthma, rheumatoid arthritis, eczema, immunosuppression); most common
- Pituitary adenoma (benign)
 - Leads to excess adrenocorticotrophic hormone (ACTH), adrenal cortisol secretion
- Adrenal Cushing's disease
 - Adrenal gland tumors/hyperplastic adrenal glands/nodular adrenal hyperplasia of adrenal glands produce excess cortisol
- Ectopic ACTH
 - Increased ACTH secreted from benign bronchial carcinoid tumors/malignant oat-cell carcinoma

COMPLICATIONS

- Metabolic syndrome, diabetes, infection due to immunosuppression, fragility fractures due to osteoporosis

SIGNS & SYMPTOMS

- Fat redistribution due to glucose release → insulin release
- Muscle, bone, skin breakdown due to protein breakdown
- Hypertension due to corticosteroids cross-reacting with mineralocorticoid receptors
- High cortisol → feedback inhibition of GH-releasing hormone (GRH) → disrupts ovarian, testicular function



Figure 11.2 Abdominal striae in an individual with Cushing's syndrome.



MNEMONIC:

BAM CUSHINGOID

Signs & symptoms of Cushing's syndrome

Buffalo hump: fat redistribution

Amenorrhea

Moon face: fat redistribution

Psycho/agitation: previously, **C**razed

Ulcers

Skin changes: acne, purple striae/stretch marks

Hypertension

Infection: due to immunosuppression

Necrosis of femoral head

Glaucoma

Osteoporosis: causing fragility fractures

Immunosuppression

Diabetes

DIAGNOSIS

DIAGNOSTIC IMAGING

CT scan

- Adrenal glands (primary Cushing's)
- Chest, abdomen, pelvis (ectopic ACTH production)

MRI

- Pituitary gland (Cushing's disease)

LAB RESULTS

- ↑ free cortisol in 24-hour urine sample
- **Cortisol blood/saliva test:** ↑ cortisol
 - Performed in evening when cortisol levels are normally low

OTHER DIAGNOSTICS

Dexamethasone suppression test

- Differentiates endogenous, exogenous Cushing's syndrome
- Measure cortisol change after dexamethasone (exogenous steroid)
- **Endogenous Cushing's syndrome:** cortisol unchanged, negative feedback cycle broken by autonomous endocrine tumor in pituitary, adrenal, etc. (ectopic ACTH)
- Positive dexamethasone test
 - **High ACTH:** ACTH-producing tumor
 - **Low ACTH:** adrenal tumor, causing pituitary ACTH suppression

Long dexamethasone suppression test

- If high ACTH
- Differentiates ACTH-producing pituitary tumor, ectopic ACTH-producing tumor (e.g. small cell lung cancer)
 - **Cushing's disease (pituitary adenoma):** cells partly responsive to negative feedback → cortisol decrease
 - **Ectopic ACTH-producing tumor:** no negative feedback → cortisol unchanged

TREATMENT

MEDICATIONS

- Cortisol inhibitors
 - Esp. if surgery ruled out by ectopic ACTH production/metastatic adrenal carcinoma
- Wean steroid medications
 - For iatrogenic Cushing's
 - Sudden withdrawal → adrenal crisis

SURGERY

- Transphenoidal resection of pituitary gland
 - For Cushing disease
- Surgical resection
 - For adrenal adenoma



Figure 11.3 A large fat deposit at the upper back in an individual with Cushing's syndrome.

HYPERALDOSTERONISM

osms.it/hyperaldosteronism

PATHOLOGY & CAUSES

- Adrenal gland produces **excess aldosterone** → hypertension (high blood pressure), hypokalemia (decreased blood potassium)
- Increased aldosterone → sodium, water retention → increased blood volume → hypertension

TYPES

Primary

- **Idiopathic** (2/3 of cases): overproduction from both adrenal glands
- **Conn's syndrome** (1/3 of cases): benign adrenal tumor → excess aldosterone
- **Familial hyperaldosteronism**: rare genetic condition, adrenocorticotropic hormone (ACTH) → adrenal aldosterone, renin secretion

Secondary

- Hypotension (e.g. congestive heart failure, cor pulmonale, hypoalbuminemia, cirrhosis, ascites, coarctation of aorta) → renin-

angiotensin-aldosterone axis activation → hyperaldosteronism

- Decreased blood flow to kidneys (e.g. renal stenosis)
- Renal-secreting neoplasms

COMPLICATIONS

- Hypertension, hypokalemia
 - Heart disease (ischemic heart disease, arrhythmias), vascular disease, renal disease, stroke, alkalosis (due to increased hydrogen ion excretion)

SIGNS & SYMPTOMS

- Headache, facial flushing (due to hypertension)
- Constipation, muscle weakness, arrhythmias (if severe, due to hypokalemia)

DIAGNOSIS

LAB RESULTS

- Renin, aldosterone levels in blood and urine
- Primary
 - Increased aldosterone, decreased renin; potassium decreased/normal
 - Metabolic acidosis secondary to hypokalemia
- Secondary
 - Increased renin, aldosterone in blood

OTHER DIAGNOSTICS

- Increased blood pressure

TREATMENT

- Goal: prevent complications of hyperaldosteronism on organs (e.g. ventricular hypertrophy, heart failure, stroke, myocardial infarction, atrial fibrillation, metabolic syndrome)

MEDICATIONS

- Potassium-sparing diuretic/aldosterone antagonist
 - Spironolactone
- Additionally
 - Thiazide diuretics, angiotensin converting enzyme inhibitors (ACE) inhibitors, calcium channel antagonists, angiotensin II blockers

OTHER INTERVENTIONS

- Control BP via lifestyle
 - Sodium restriction, weight management, regular exercise
- Second-line
 - Thiazide diuretics, ACE inhibitors, calcium channel antagonists, angiotensin II blockers